

of cardiac degeneration, those with a regular rhythm, and the tachycardia of fever do not respond so well. Lewis considers it due to a partial block, and that cases with a prolonged A-C interval, that is, with a delay in conductivity, will become slower on digitalis. But clinically, the cases with rapid irregular action, that is, with a short conduction time, become slower. Cushing thinks that digitalis increases inhibitory action of the vagus and prevents many impulses reaching the ventricle. Is the action mainly vagal, or are the conducting fibers of the auriculo-ventricular bundle themselves, or is the heart muscle lowered in irritability due to improved nutrition? In the senile type there is loss of inhibitory power of the vagus, and so atropine has little effect; also the cardiac musculature is so degenerated that nutrition cannot improve it much. By action of atropine the effects of vagus inhibition could be removed and so one of the possible factors would be cut out. The patient's pulse was studied at rest, under atropine alone, under digitalis, and under the drugs combined. It was found in the rheumatic cases with rapid heart that the rate was slowed by digitalis, and on administration of atropine, while the rate was increased, it in no case reached the original rate before drugs; if the effect had been due solely to vagal inhibition, it is fair to suppose that following atropine the old rate would have been reached. There are thus two component actions of digitalis, one vagal and the other on cardiac tissue, and they vary in different conditions. In the cases of auricular fibrillation that give high increases of pulse rate on the administration of atropine, digitalis works its best, for following digitalis atropine has almost no effect in raising the rate; that is, most of the effects of the digitalis are on the cardiac musculature. These cases, as a rule, do well and may even resume work again. By the study of pulse rates following atropine with and without digitalis, it is possible to find out whether digitalis is acting upon vagus or cardiac tissue.

Pathology of the Dyscrasic Edemas.—DÉBOVE (*Presse Méd.*, 1911, xix, 517). While edema due to mechanical cause has long been differentiated from that due to a chemical change in the blood, it is well to remember that the blood composition is kept constant by the kidneys. An excess of salt or water is quickly eliminated in health by the organs acting as a selective filter and removing the superabundant ingredient. But the excretion of a concentrated toxic fluid by the kidney may endanger the organ. So in the diseased body it is usual to find the superabundant toxins distributed not alone in the blood, but in the general body fluids. For example, if one ligates the ureters and then injects a rabbit with some dyestuff, this material will not be recoverable from the blood, but from the general body. As the toxins can only remain in the body fluid in solution, they are diluted and result in edema, which thus appears to be a defensive reaction against toxins, and preserves the chemical constitution of the blood intact. In typhoid fever and pneumonia, for example, the patient loses little in weight in spite of high fever, because of edema that is not recognizable because it is so general. In the diuresis of convalescence the toxins are rapidly poured out by the kidneys and weight is lost. This rapid outpouring of toxins could, in pneumonia, be spoken of as a chloruria, because chlorides had been retained to make the toxic solution physiologically

normal and are now excreted rapidly. Not only does edema save the kidney from excreting too highly toxic fluids, but as antibodies are present in all transudates, the toxins are catabolized in the tissues and so rendered innocuous. Probably the pathogenesis of edema is the same in nephritis. Nephritis is largely due to a hematogenic infection. Frequently the fact is overlooked that the causative agent persists even if the kidney lesion makes no progress. Here the organism protects itself from the assault by edema in hydropic nephritis and by polyuria in the anhydropic. Clinically, in the hydropic form, it has been observed that a disappearance of edema has been followed by an exacerbation of the symptoms caused originally by the toxins, a fact that can be explained only by supposing the elimination of a too highly concentrated urine. But while Widal and Javal believe the edema of nephritis to be due to a primary retention of chlorides through kidney impermeability, Débove thinks it a primary retention of toxins. The blood and lymph contain the same amounts of chlorides before, during, and after nephritis, and it is highly probable that water and salts are retained to make the toxins dilute enough, also isotonic. When salt is excluded from the diet the edema is reabsorbed and gradually excreted. Sodium chloride is one of the simplest things for the kidney to excrete in health. In disease this power is somewhat impaired, but by no means so much as is the power of urea excretion. He thinks of flooding the kidney with a highly toxic urine. In this toxicity there is no reason to believe that sodium chloride plays any important part. In the interstitial form the organism tries to secure elimination by polyuria because the toxins are different and have acted upon different kidney structures. While many types of edema are mechanical in origin, we cannot afford to overlook the primary chemical change in the blood as is found in cirrhosis of the liver.

Functional Diagnosis of the Liver.—F. FALK AND P. SAXL (*Zeit. f. klin. Med.*, 1911, lxxiii, 131) point to the fact that while amino-acids have frequently been found in the urine in large quantity in hepatic disease, practically no attention has been paid to the occurrence of the more complex nitrogenous bodies (polypeptids). The authors, therefore, undertook a study of the amount of amino-acid nitrogen and of peptid nitrogen in cases of primary liver disease and also in cases where the liver was affected secondarily. To determine the peptid N, they used the formalin titration method of Sørensen. As Falk and Saxl have previously shown, this method, as modified slightly by them, gives results which, though not absolute, are sufficiently accurate for clinical purposes. The studies of Falk and Saxl show that hepatic disease in general causes an increase in the relative quantity of both the amino-acid and the peptid N. Ordinarily the two are increased proportionally, but exceptions to this are quite frequent. After feeding glycocholl, they find no changes in the nitrogenous fractions in patients with normal livers; with hepatic disease, and particularly cirrhosis, they observed a great increase in the peptid N fraction.

The Blood Findings in Epidemic Cerebrospinal Meningitis.—RUSCA (*Deut. Archiv f. klin. Med.*, 1911, ciii, 235) has made a rather complete study of the blood in nine cases of epidemic cerebrospinal meningitis,